Surgical management of Cerebral Aneurysms

by

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No disclosure that would be a potential conflict of interest with this presentation
Plan:

- Epidemiology
- Etiology
- Histology of intracranial arteries
- Location
- Presentation of aneurysms
- Conditions associated with aneurysms
- Classic saccular aneurysm: pathogenesis, risk factors, vessel wall pathology, histopathology
- Surgical approaches to aneurysms
- Surgical technique
- Cases of brain aneurysm
Epidemiology

Difficult to estimate

Autopsy prevalence: 0.2 – 7.9%, it depends

Prevalence 5%

Ratio of ruptured: unruptured 5:3 to 5:6
(rough estimate is 1:1)

50% of these aneurysms rupture

Only 2% present during childhood
Etiology

- Congenital predisposition (medial gap/muscular layer)
- Atherosclerotic or hypertensive
- Embolic: as in atrial myxoma
- Infectious (mycotic aneurysm)
- Traumatic
- Associated with other conditions
Histology of intracranial arteries

- **Intima** (17%), **media** (52%) and **adventitia** (31%)
- Endothelium and subendothelial connective tissue
- Internal elastic lamina (mechanical strength) separates the I and M
- M consists of closely packed layers of smooth muscle cells surrounded by collagen and elastin fibers
- No external elastic lamina between M and Ad
- Ad is very thin
- Lower wall:lumen ratio
- Mean wall thickness 0.6 mm in the Basilar and 0.5 mm MCA
- Intradural arteries are surrounded by CSF remain in this state after entering the brain parenchyma (perivascular space/Virchow-Robin spaces)
- Ensheathed by “glia limitans” -> specialized layer of astrocytes
Location

- Apex of branch points which is the site of max hemodynamic stress
- More peripheral do occur associated mycotic aneurysm or trauma
- Fusiform aneurysms more common in the VertBasilar system
- Dissecting aneurysm (categorized with arterial dissection)
- Saccular aneurysm locations
  - 85-95% in the carotid system: AcoA 30% p-com 25% MCA 20%
  - 5-15% in the posterior circulation: 10% basilar tip 5% vertebral
- 20-30% have multiple aneurysms
Presentation

• Rupture
  • SAH
  • Intracerebral hemorrhage 20-40%
  • IVH 13-28%
  • Subdural hematoma 2-5%
opening in the subarachnoid space of the brain created by a separation of the arachnoid and pia matter
Intraventricular hemorrhage

• 13-28%
• Prognosis worse (64% mortality)
• The size of the ventricle is the most important prognosticator
• Acom aneurysm -> rupture through the lamina terminalis -> anterior 3rd ventricle or lateral ventricle
• Basilar tip or carotid terminus aneurysm -> floor of the 3rd ventricle
• Distal PICA -> into the 4th ventricle via foramen of Luschka
Other presentations

• Mass effect: giant aneurysm
  • Brainstem compression -> hemiparesis and cranial neuropathies
  • Cranial neuropathies
    • Non-pupil sparing third nerve palsy Pcom
    • Visual lost:
      • compressive optic neuropathy carotid ophthalmic aneurysm
      • Chiasmal syndrome ophthalmic, acom, basilar tip
      • Facial pain syndrome (ophthalmic/maxillary nerve distribution) intracavernous

• Intra or suprasellar aneurysm -> endocrine disturbance due to pituitary compression
Other presentations

- Minor hemorrhage: warning or sentinel
- Small infarct or TIA: distal embolization
- Seizure: localized gliosis
- Headache without hemorrhage
  - acute (expansion, thrombosis or intramural bleed)
  - > 2 weeks irritation of overlying dura, mass effect -> increase ICP
- Incidental discovered (asymptomatic)
Conditions associated with aneurysms

- Autosomal dominant polycystic kidney disease
- Fibromuscular dysplasia (abnormal growth within the wall)
- AVM
- Connective tissue disorders: Ehlers-Danlos type 4 (deficient collagen type 3), Marfan’s syndrome, pseudoxanthoma elasticum
- Multiple other family members with intracranial aneurysms
- Coarctation of the aorta
- Osler-Weber-Rendu syndrome (Hereditary hemorrhagic telangiectasia)
- Atherosclerosis
- Bacterial endocarditis
Polycystic kidney disease

- Transmission is autosomal dominant
- Progressive chronic renal failure
- HTN is a common sequelae
- Prevalence of aneurysms with ADPKD is 10-30% around 15%
- Increased incidence, increased risk of rupture
- 10-20 fold increased risk of SAH compared to general population
- Average rate of rupture of incidental aneurysm is 2%/year
- Screening with MRA is beneficial
Classic saccular aneurysm

- Focal outpouching of the wall
- Site of bifurcation
- Pointing in the direction of the blood flow
- Berry-like morphology
- Prevalence is 3.2% (for unruptured)
Pathogenesis

• not completely understood
• Congenital disease (historically, assumption is incorrect, acquired)
• Caused by
  • hemodynamic stresses (luminal factors)
    • Supported by preferred location (bifurcation) and increase association with arterial anatomical variants (agenesis, persistent anastomoses, asymmetry and fenestrations)
  • defective vessel wall responses (abluminal factors)
• Exogenous factors are associated with formation and rupture
  • Cigarette smoking
  • Alcohol consumption

• Genetic foci associated with the development of aneurysms
  • Chromosomes 18q11.2, 10q24.32, 8q11.23-q12.1, 9p21.3
  • Explain only up to 5% of familial risk of intracranial aneurysm
  • The currently available genetic risk prediction tests are not well-established screening methods
Risk factors for aneurysm formation and rupture

- Female
- Increasing age
- HTN
- Tobacco smoking
- Excessive alcohol consumption
- Family history of aneurysm
- Polycystic kidney disease
- Previous SAH
Vessel wall pathology

- Histopathological feature: disruption of the internal elastic lamina
- Tears in IEL (induced by alteration of hemodynamic)
- Smooth muscle cells
  - can migrate into the intima
  - Proliferate -> myointimal hyperplasia (adaptive response)
- Endothelium composed of either,
  - near normal pattern of smooth and linearly organized endothelial cells
  - or irregular surface formed by increased endothelial-cell apoptosis and adhesion of blood cells
- Media composed of dense or disorganized layers of smooth muscle cell or near absence of smooth muscle cells
- Adventitia composed of collagen fibers, may be stretched
luminal subacute thrombus, a pattern of alternating thinning and thickening of the vessel wall, lack of smooth muscle cells (arrows) in a large portion of the wall, and a mild infiltrate of adventitial lymphocytes (arrowheads). Loss of the internal elastic lamina
Unruptured vs Ruptured aneurysm

• Unruptured aneurysm
  • Intact endothelium
  • Intact smooth muscle cell layers
  • Few macrophages and other inflammatory cells

• Ruptured aneurysm
  • Disrupted endothelium
  • A few smooth muscle cells
  • Diffuse invasion of macrophages and leukocytes

Aneurysm-wall inflammation is present before the rupture occurs and is not a result of the rupture
Classification of aneurysms by their wall histopathology (structural changes)

• Orderly layers of smooth muscle and intact endothelium

• Disorganized layers of smooth muscle

• decreased numbers of smooth muscle cells and myointimal hyperplasia or organized thrombus

• Thin hypocellular wall and endothelial-cell apoptosis
Microarray gene analysis

found differential gene expression profiles between healthy wall and aneurysm wall

• Increased expression of genes related to
  • Infiltration of inflammation cells
  • Complement activation
  • Apoptosis
  • Inhibition of re-endothelialization in the aneurysm wall

Both structural changes and an inflammatory response within the aneurysm wall seem to precede aneurysm rupture.
Surgical approaches to aneurysms
most common sites of saccular aneurysms
Lateral and superior view of common aneurysms on the supraclinoid portion of ICA
Operative view of aneurysm sites on the ICA
Fronto temporal (pterional) craniotomy

Used to expose aneurysms on the Circle of Willis
Perforating arteries at common aneurysm sites
Approach to PCOM
Approach to PCOM
Approach to MCA
Microinstruments for microsurgery
Blunt dissection vs sharp dissection
Approach to ACOM
Distal part of the Anterior cerebral artery

Interhemispheric approach
Common aneurysm
Sites in the posterior fossa
Surgical technique
Fig. 15.4 Dissection steps in splitting the sylvian fissure (cerebral and superficial dissection, right side). Step 1: cortical arachnoid reflection; step 2: temporal mobilization of the sylvian veins.

Fig. 15.3 Normal variations of the superficial sylvian vein anatomy (right side): (A) Abnormal superficial sylvian veins, (B) Single superficial sylvian vein, (C) Parallel superficial sylvian veins, (D) Complex network of superficial sylvian veins.
- Indocyanine green: fluorescent dye
- intra operative angiogram
- intra operative doppler
Cases of brain aneurysm
69 years-old female

PAST MEDICAL HISTORY: DVT, HTN, Diabetes

Rx: coumadin,


P/E:
Right ptosis
PERL, pupils normal size
Ocular mvt normal
No drift
Strength 5/5, DTR 2/4, no sensory deficit to pin prick

Blood work: INR 5.5
Treament: coiling

The International Subarachnoid Aneurysm Trial (ISAT) demonstrated that patients with ruptured aneurysms rated by both the neurosurgeon and the interventional neuroradiologist as potential candidates for therapy had lower morbidity, dependency and mortality rates when treated with endovascular coiling than when treated with neurosurgical clipping.
Repositioning of the first coil
Second coil
2mm x 1.5 cm
Final result
After removing the microcatheter
Pipeline
What is Pipeline?

Flexible mesh like device

Bimetallic self expanding braid

25% platinum tungsten
75% cobalt chromium

48 strands interwoven in standard pattern

30-35% surface coverage
Low porosity stent → Redirecting blood flow → Intra aneurysmal stasis → Thrombosis within the aneurysm → Subsequent endothelialization → Excluded aneurysm
Conclusion:

.Aneurysms are unique lesion
.Individualized mixture of geometry, size, location, relationship to surrounding
.lead to devastating morbidity and mortality:
  high risk of rupture associated with
    posterior circulation and PCOM
    size> 7mm
    high aspect ratio or bottle neck
    irregular surface and daughter sac
    small parent artery
.Challenging to treat
.Many options to offer the patients